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Cortisol as a Moderator of Acute Distress in Predicting PTSD

Hannah L. Ward

College Honors Thesis

Department of Psychological Science

University of Vermont

### Abstract

Following traumatic injury, approximately one third of patients admitted to the hospital will develop a psychiatric condition, including posttraumatic stress disorder (PTSD; O'Donnell, Bryant, Creamer, & Carty, 2008). It is unknown, however, why some develop PTSD and others recover. Theorists have proposed acute distress as a potential factor that contributes to identifying PTSD risk. Cortisol is a widely used marker of stress reactivity, and prior work has shown that people with inhibited cortisol reactions following trauma are at higher risk for subsequent PTSD (Price, Kearns, Houry, & Rothbaum, 2014). This project investigated the interaction between the acute stress reaction and cortisol with regards to their relation to subsequent PTSD symptoms. No significant interaction was found between cortisol and later PTSD, with the null result likely stemming from measurement related confounds. The data suggest that salivary cortisol collected shortly after the trauma in conjunction with acute distress may be an unreliable biomarker of PTSD.

### Cortisol as a Moderator of Acute Distress in Predicting PTSD

The majority of US adults will be exposed to a traumatic event in their lifetime (Kilpatrick, Resnick, Milanak, Miller, Keyes, & Friedman, 2013). Although many recover, a substantial minority will develop chronic mental health problems including posttraumatic stress disorder (Bryant, O'Donnell, Creamer, McFarlane, Clark, & Silove, 2010; Zatzick, Rivara, Nathens, Jurkovich, Wang, Fan, & Mackenzie, 2007). Understanding the mechanisms associated with resilience, recovery, or chronic mental illness is necessary in order to create effective prevention strategies. After being admitted to the Emergency Department (ED) following traumatic injury, physicians work to screen patients for injuries and proceed with a specific course of treatment. Despite the fact that approximately one third of traumatic injury patients will develop a psychiatric condition, there are few screening or referral mechanisms in place for these conditions that have been widely used outside of research (O'Donnell et al., 2008). Due to this lack of early detection, many patients who develop PTSD as a result of their traumatic experience will not receive mental health treatment. Indeed, epidemiological studies indicate that two thirds of patients who develop post-trauma mental health conditions do not receive treatment (Bryant et al., 2010).

There are a number of factors associated with post-trauma psychopathology including physiological, hormonal, psychosocial, and environmental variables. Although these variables show promise as predictors individually, no single construct has emerged as a reliable and valid predictor of PTSD (for a review, see Bryant, 2006). It is highly likely that the development of PTSD is influenced by multiple factors that interact with one another. The primary aim of the current study is to improve our understanding of the complex interplay of factors that contribute to subsequent PTSD. Salivary cortisol and acute distress have both been individually associated

with the development of PTSD, and the extent to which they interact with one another to predict PTSD was examined.

The proposed factors in the current study have been identified as part of the construct of fear load. Fear load is a multidimensional construct defined as the over-expression of a fear response when exposed to a trauma cue and is influenced by many factors including symptom severity at the time of the event, acute distress following exposure to a stressor, physiological response, dissociation, and genomics (Norrholm, Glover, Stevens, Fani, Galatzer-Levy, Bradley, Ressler, & Jovanovic 2014). Norrholm and colleagues found higher fear load in populations that are both extremely symptomatic and in most need of treatment, thus supporting a relationship between fear load and PTSD. Notably, recent research suggests that a highly sensitized fear response is most active and significantly correlated with intrusive thoughts and intense physiological reactions to reminders of the trauma, both of which are characteristic symptoms of PTSD as defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; APA, 2013). This project will examine two components of the fear load construct by focusing on acute distress and post-trauma physiology as measured by cortisol.

Acute stress at the time of the trauma has been associated with subsequent PTSD symptoms (Birmes, Brunet, Benoit, Defer, Hatton, Sztulman, & Schmitt 2005; Nishi, Matsuoka, Yonemoto, Noguchi, Kim, & Kanba, 2010). The severity of this acute distress reaction is of particular importance because it can be more easily assessed in an acute care setting. Such assessments can then guide an immediate course of clinical action. Unfortunately, acute stress has been shown to be unreliable as a predictor of PTSD when examined alone. Indeed, studies that have shown a unique relation also included additional variables in the model such as peritraumatic dissociation, heart rate, and other physiological variables (Kuhn, Blanchard, Fuse,

Hickling, & Broderick, 2006). These results suggest that acute distress may be most useful as a predictor when used in conjunction with other variables.

The present study will also examine one of the physiological responses that occurs as a result of the acute reaction. Although the degree of activation may vary depending on the situation, experiences of traumatic events initiate a response of the hypothalamic-pituitary-adrenal axis (HPA; Hellhammer, Wüst, & Kudielka, 2008). During an acute stress response and subsequent activation of the HPA axis, glucocorticoids, namely cortisol, are produced in the adrenal cortex (Meewisse, Reitsma, de Vries, Gersons, & Olf, 2007). The release of cortisol terminates the stress response through a negative feedback loop and as such one of its major functions is to contain the stress reaction (Yehuda, McFarlane, & Shalev, 1998). Cortisol levels are thus generally considered to be a reliable measurement of HPA activity, however, certain sources of variability are acknowledged. Prior trauma exposure, for example, has been shown to attenuate the acute cortisol reaction in response to subsequent traumas, as supported by the results of a study that found women with a previous history of assault tended to display lower mean cortisol levels after rape (Resnick, Yehuda, Pitman, & Foy, 1995). The lowered cortisol response was associated with a higher probability of subsequent PTSD.

Acute distress and cortisol may thus be important factors to examine in a combined model as predictors of PTSD symptomology. The present study aims to understand the complex relationship between acute distress and the cortisol response, and their association with subsequent PTSD in the acute aftermath of a trauma. Elevated cortisol has been identified as a potential protective factor against PTSD symptoms, and as such was examined as a moderator of the association between acute distress and PTSD. Specifically, it was hypothesized that (1) acute distress would be positively associated with PTSD symptoms, (2) cortisol would be negatively

associated with PTSD symptoms, and (3) that there would be an interaction between cortisol and acute distress such that elevated cortisol would reduce the strength of the relation between acute distress on PTSD symptoms.

## **Methods**

### **Participants**

Patients who presented to the Emergency Department of The University of Vermont Medical Center immediately following a trauma were screened for eligibility in the study through cooperation with the Emergency Medicine Research Associate Program (EMRAP) and Trauma Center staff. Patients were also recruited through the UVM Medical Center Trauma Clinic. Patients who presented after an event meeting Criterion A for the DSM-5 diagnosis of PTSD were approached for enrollment (APA, 2013). Patients were excluded based on current suicidal ideation, current psychoses, being in police custody, and/or altered state of mind as determined by the attending physician. A total of 72 participants were enrolled.

Participants in the current sample were  $M = 35.15$ ,  $SD = 13.91$  years of age. The majority of the sample self-identified as White (67.2%) and approximately one fourth of the sample identified as African American (26.9%). The majority of the sample was male (64.2%). Most participants identified that they had private insurance (41.8%).

### **Measures**

The following measures were administered to enrolled participants.

**Peritraumatic Distress Inventory (PDI;** Brunet et al., 2014). The PDI is a measure aimed at quantitatively measuring the amount of distress experienced as a direct result of a traumatic experience. Participants completed this 13-item self-report measure, rating each item on a scale of 0-4 relative to the amount of distress experienced at the time of trauma; total scores

ranged from 0 to 52 with higher scores being representative of greater distress. Research indicates that the PDI is an accurate predictor of post-traumatic stress symptoms and PTSD at follow-up several days after the traumatic event (Nishi et al., 2010).

**Peritraumatic Dissociative Experiences Questionnaire** (PDEQ; Marshall et al., 2002).

The PDEQ is a 10-item self-report measure designed to assess of the amount of dissociation experienced at the time of trauma. Items are rated on a scale of 1-5 based on extent of dissociation experienced with total scores ranging from 10-50 and higher scores indicating more dissociation. Psychometric analyses have found that the PDEQ total score is significantly correlated with other measures of acute dissociation and thus finds peritraumatic dissociation to be a valid predictor of subsequent PTSD symptomology or diagnosis (Birmes et al., 2005).

**PTSD Checklist-5** (PCL-5; Weathers et al. 2013). The PCL-5 is a 20-item self-report measure that assesses PTSD symptoms in the last month according to the DSM-5 criteria. Items assess symptoms across the four symptom clusters of PTSD (re-experiencing, dysphoria, avoidance, and hyper arousal) on a 0-4 Likert scale. Total scores range from 0-80, with higher scores indicating increased symptomology.

**Procedure**

Participants were recruited through the ED at The University of Vermont Medical Center, a Level 1 Trauma Center. EMRAP and Trauma Center staff reviewed each admission log in order to determine patient eligibility, and eligible candidates were approached for enrollment. If enrolled, participants completed the aforementioned measures and provided a salivary cortisol sample for analysis. Cortisol samples were collected with a Salimetrics SalivaBio Oral Swab from Salimetrics, LLC (Carlsbad, CA) that was placed underneath the participant's tongue for 1-



2 minutes. Swabs were stored in the EMRAP -80°F freezer following collection until which time all were sent to Salimetrics for analysis.

Follow-up phone calls were completed by the Center for Research on Emotion, Stress, and Technology at the University of Vermont and all measures were re-administered at 1-week, 1-month, and 3-months post-trauma.

### Results

Of the 72 cortisol samples obtained, 5 were excluded due to extreme values ( $> 1.5$   $\mu\text{g/dL}$ ). These extreme values were attributed to contaminated samples in which blood was included on the swab, thus artificially inflating the cortisol value. The remaining analyses were conducted with a sample of  $n = 67$  participants. Cortisol values were  $M = 0.21$   $\mu\text{g/dL}$ ,  $SD = 0.16$ . Next a series of ANOVAs determined that there were no significant differences in cortisol values between variables that may have affected cortisol readings (Table 1). The relation between time of day and cortisol samples approached significance and was used as a covariate in cortisol analyses,  $r = -.21$ ,  $p = .09$ . This relation is consistent with prior work that shows cortisol levels naturally decline over the course of the day.

The association between peritraumatic stress and PTSD symptoms was evaluated with regressions (Table 2). Week 1 PTSD symptoms were used as a covariate when evaluating PTSD symptoms at Month 1 and Month 3. There was a significant relation between peritraumatic distress and PTSD symptoms at Week 1 ( $b = 1.05$ ,  $p < .01$ ) in the hypothesized direction. After controlling for Week 1 PTSD symptoms, the relation between PTSD at Month 1 and peritraumatic distress was significant but not in the hypothesized direction ( $b = -0.67$ ,  $p < .01$ ). This significant negative relation is attributed to the high correlation between PTSD symptoms at Week 1 and Month 1 ( $r = .73$ ,  $p < .01$ ), which resulted in a suppression effect. The bivariate

relation between peritraumatic distress and Month 1 PTSD symptoms was in the expected direction, but did not attain significance ( $b = 0.43, p = 0.08$ ). Finally, the relation between peritraumatic distress and PTSD at Month 3 was not significant ( $b = -0.32, p = .25$ ).

The association between cortisol and PTSD symptoms was next examined using a regression analysis. Time at which the cortisol sample was obtained was included as a covariate for all analyses. Cortisol was not found to be associated with PTSD symptoms at Week 1 ( $b = 12.28, p = .39$ ) or Month 1 ( $b = .08, p = .99$ ). Cortisol was also not associated with PTSD symptoms at Month 3 ( $b = 2.05, p = .88$ ). Taken together, these findings suggest that salivary cortisol was unrelated to subsequent PTSD symptoms.

Despite the null relations, the potential moderating effect of cortisol on the association between acute distress and subsequent PTSD symptoms was explored. The interaction between peritraumatic distress and cortisol on Week 1 PTSD symptoms was not significant ( $b = -0.12, p = 0.89$ ). The interaction between peritraumatic distress and cortisol on Month 1 PTSD symptoms was not significant ( $b = 0.27, p = 0.73$ ), nor was the interaction at Month 3 ( $b = 0.89, p = 0.39$ ). These data suggest that cortisol and peritraumatic stress do not interact to alter PTSD symptoms.

### **Discussion**

The present study investigated the association between acute distress, salivary cortisol shortly after a trauma, and subsequent PTSD symptom severity. Despite compelling evidence from prior work, the current study did not support the hypothesized set of relations. No relationship was found to exist between cortisol levels and PTSD symptoms at subsequent time points. Acute distress was associated with PTSD symptoms shortly after a trauma, but this relation was no longer significant when examining PTSD symptoms in the months after the trauma. Furthermore, the moderating relationship between acute distress, salivary cortisol, and

PTSD symptoms was not significant. Taken together, these results call into question the utility of using salivary cortisol and acute distress shortly after a trauma as a predictive biomarker of PTSD symptoms.

The results of the present study contrast with prior research that has shown reduced cortisol levels after trauma to be associated with subsequent PTSD symptoms. There are several potential explanations for this discrepancy in findings. The use of cortisol as a predictor of PTSD relies on its ability to be both sensitive and specific in identifying those at high risk; however, research demonstrates salivary cortisol may be unreliable when used diagnostically in this manner. Salivary cortisol levels fluctuate over the course of the day. Indeed, research indicates that free salivary cortisol levels increase by 50-75% within the first half hour after waking in the morning (Pruessner et al., 1997). The present study collected samples at the time at which there was access to the patients and, as a result, was unable to control for time of day. The time of day of sample collection may have thus affected the level of cortisol obtained and the strength of the relation. Second, although sample collection occurred as close as possible to the traumatic event, there was still a great deal of variability in time from trauma. This variability was attributed to limitations in approaching certain participants, or the time at which they presented to the ED. This variability may have further reduced the strength of the relation between cortisol and PTSD. Prior work in which a significant relation was found reported sample collection consistently closer to the time of the trauma (e.g., Resnick et al., 1995, Meewisse et al., 2007; Yehuda et al., 1998). Specifically, sample collection occurred within hours of the event in these studies.

Trauma history is also an important consideration in future research, as an attenuated cortisol response in those with prior traumatic exposures has been observed. Specifically, Resnick and colleagues (1995) found that only women with no history of severe sexual assault

had higher cortisol levels following high-severity rape, as compared to women with a history of previous trauma. This may indicate the use of cortisol as a biomarker of PTSD only in cases of those without prior trauma exposure. The current study was not able to control for prior trauma exposure, which may have affected the results. Similarly, recent work has suggested that the timing and severity of traumatic events is more relevant to recovery than mere exposure to such events (Teicher & Parriger, 2015). It may be the case that the current sample had less frequent and severe trauma exposure than other samples, which limited the strength of the relation.

Furthermore, the present study assessed cortisol levels only at a single time point. Prior research demonstrates that cortisol levels taken over time tend to correlate with PTSD symptom severity (Olf, de Vries, Güzelcan, Assies, & Gersons, 2007). Studies that suggest an association between increasing cortisol levels over time and improvement in PTSD symptomology would also support the use of a longitudinal assessment of cortisol (e.g. Kellner, Yehuda, Arlt, & Wiedemann, 2008). Analysis utilizing an average of multiple cortisol samples as opposed to one taken at a singular time point may prove lower overall as compared to healthy controls (Olf, Güzelcan, de Vries, Assies, & Gersons, 2006). Average cortisol levels taken overtime may thus be a more reliable indicator of acute distress and PTSD.

The current study had several limitations of note. PTSD symptomology was assessed using exclusively self-report measures, which may have limited the accuracy of the symptom measurements. Other studies have used diagnostic interviews that are thought to have high reliability and validity (Foa, Cashman, Jaycox, & Perry, 1997). The present study also included a heterogeneous trauma sample whereas other studies used a sample that experienced a relatively homogenous event (e.g., sexual assault only). Indeed, Hellhammer and colleagues (2009) suggested that although endocrine functions are generally covariate with psychological stress

responses, this relationship is inconsistent when examined across different stressors and diverse subject populations. Further investigation of cortisol and acute distress in a more homogenous population of trauma may yield different results. Specifically, the corticosteroid response may be stress specific, which would thus alter the relations in a heterogeneous sample. A recent study in rodents indicates that differences in the type of stress exposure may affect physiological responses differently in that physically demanding and life threatening stimuli result in an increased adrenal response whereas psychological, anxiety-inducing stressors result in a decreased response (Bornstein, Engeland, Ehrhart-Bornstein, & Herman, 2008). This would support a research design investigating only a particular subset of trauma patients rather than a varied sample, as varying traumas may differently affect the cortisol response.

Despite the lack of significant relation found between cortisol and acute distress in the present study, there is a benefit to understanding the biological mechanisms that increase risk for post-traumatic stress. Although cortisol collection shortly following trauma was shown to be an unreliable indicator of PTSD in this sample, future research should continue to investigate combinations of biomarkers in order to develop clinically useful screening tools for trauma exposed populations.

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Table 1. *ANOVAs comparing cortisol levels across factors known to affect cortisol levels.*

	df	F	<i>p</i>
Ate in the last hour	1, 60	0.004	0.95
Tobacco use last 12 hours	1, 60	0.713	0.4
Alcohol use last 12 hours	1, 60	0.324	0.57
Currently using birth control	1, 60	0.42	0.52
Currently pregnant	1, 60	0.05	0.83

Table 2. *Regressions of associations between cortisol and acute distress*

<b>Dependent</b>						
<b>Variable</b>	<b>Predictors</b>	<b>B</b>	<b>SE</b>	<b><math>\beta</math></b>	<b>t</b>	<b>p</b>
Week-1 PCL	PDI	1.051	0.156	0.676	6.74	< 0.001
Month-1 PCL	PDI	-0.668	0.203	-0.423	-3.297	0.002
	Week-1 PCL	1.043	0.131	1.021	7.953	< 0.001
Month-3 PCL	PDI	-0.323	0.278	-0.205	-1.159	0.254
	Week-1 PCL	0.793	0.181	0.775	4.38	< 0.001
Week-1 PCL	Cortisol	12.28	14.124	0.118	0.869	0.389
	Time of Day	-0.009	0.004	-0.28	-2.07	0.044
Month-1 PCL	Cortisol	0.078	10.899	0.001	0.007	0.994
	Time of Day	-0.002	0.003	-0.073	-0.613	0.543
	Week-1 PCL	0.701	0.118	0.709	5.941	< 0.001
Month-3 PCL	Cortisol	2.053	13.207	0.021	0.155	0.877
	Time of Day	0.006	0.004	0.218	1.533	0.135
	Week-1 PCL	0.716	0.144	0.711	4.967	< 0.001

Note: PCL = PTSD Symptom Check List.